

Chapter 50
**Mercury (Thimersol) and Aspartame as Cofactors
in the Epidemic of Neurodegenerative Diseases**

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ABSTRACT

The artificial sweetener aspartame (6-methyl-1,2,3-oxathiazine-4[3H]—one-2,2-dioxide salt of L-phenylalanyl-2-methyl-L-alpha-aspartic acid), is consumed, primarily in beverages, by a very large number of Americans, causing significant elevations in plasma and brain phenylalanine levels. It is very likely that aspartame, which was once considered a new chemical warfare agent by the US military has resulted in an enormous toll in illness, disability, and death. The failure of the medical profession and many governmental and other public health agencies to concern themselves with this ignored epidemic parallels what has taken place with the use of Thimerosal in vaccines.

As with Thimerosal, the most grievous offense of the illegal approval and continued use of aspartame pertain to the damage that this chemical can induce in infants and children. Moreover, aspartame could affect subsequent generations born to mothers who were misled about the safety of this and related chemicals. This paper will discuss the role of both aspartame and Thimerosal in the pathology of neurodegenerative disease.

INTRODUCTION

The chemicals we ingest may affect more than our own health.

They affect the health and vitality of future generations.

*The danger is that many of these chemicals may not harm us
but will do silent violence to our children.*

Senator Abraham S. Ribicoff (1971)

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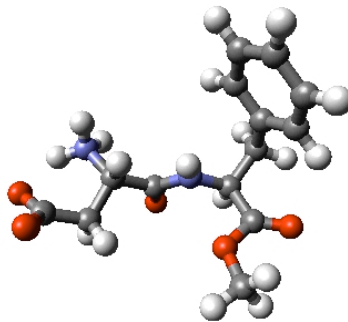


Figure 1. The aspartame molecule

THE HISTORY OF ASPARTAME

In October 1980 the Public Board of Inquiry (PBOI) impaneled by the FDA to evaluate aspartame safety found that the chemical caused an unacceptable level of brain tumors in animal testing. Based on this fact, the PBOI ruled that aspartame should not be added to the food supply.

This ruling culminated 15 years of regulatory ineptitude, chicanery, and deception by the FDA and the Searle drug company, aspartame's discoverer and manufacturer (acquired by Monsanto in 1985), and then started the ball rolling on two additional decades of maneuvering, manipulating, and dissembling by the FDA, Searle, and Monsanto.

In 1965, a Searle scientist licked some of a new ulcer drug from his fingers and discovered the sweet taste of aspartame. Searle's early tests showed that aspartame produced microscopic holes and tumors in the brains of experimental mice, epileptic seizures in monkeys, and was converted into formaldehyde.

Despite of the information in its files, in 1974 the FDA approved aspartame as a dry-foods additive. The renowned brain researcher, John Olney from Washington University in St. Louis reviewed the available data and discovered two studies showing brain tumors in rats and petitioned the FDA for a public hearing. Dr. Olney had already shown that aspartic acid (part of the aspartame molecule) caused holes in the brains of rats. Aspartame also is one part phenylalanine, and one part methyl (or wood) alcohol.

The FDA prevailed on Searle to refrain from marketing aspartame until after completion of the hearing. In 1975, an FDA Special Commissioner's Task Force reported serious problems with Searle's research that was conducted in a manner so flawed as to raise doubts about aspartame safety and create the possibility of serious criminal intent. The FDA asked the US Attorney for Chicago to seek a grand jury review of the monkey seizure study, but he let the statute of limitations run out, then (along with two aides) proceeded to join Searle's law firm.

In October 1980, the PBOI blocked aspartame marketing until the tumor studies could be explained, and unless the commissioner overruled the board, the matter was closed. In November 1980, Ronald Reagan was elected President and Donald Rumsfeld, president of Searle, joined the Reagan White House. In January 1981, Rumsfeld told a sales meeting that he would call in his chips and get aspartame approved – Dr. Arthur Hull Hayes, Jr. a pharmacologist and Defense Department contract researcher became FDA commissioner and his first decision was to defy FDA advisors and approve aspartame for dry foods. His last decision, before leaving his post because of improprieties (taking gifts from Pharmaceutical companies) was to approve aspartame for soft drinks in 1983. He immediately became senior medical advisor to Searle's public relations firm for \$1000/day. Rumsfeld received a \$12 million bonus.

As soon as soft drinks with Nutrasweet began to be consumed, complaints began to arrive at the FDA – dizziness, blurred vision, headaches, and seizures. The complaints were more serious than the FDA has ever received on any food additive. In 1985, the FDA asked the Centers for Disease Control (CDC) to review the first 650 complaints (there are now tens-of-thousands). The CDC found that the symptoms in approximately 25% of cases stopped and then restarted with discontinuing the use of aspartame and then restarting its use. The day the FDA released the CDC report, which they discounted, Pepsi Cola announced its switch to aspartame with a worldwide media blitz.

At the same time, human brain tumors rose 10% and previously benign tumors turned virulent. An FDA deputy commissioner said the data posed no problem; he then became Vice President of clinical research for Searle.

Four hundred aspartame studies were done between 1985 and 1995. All of the studies Searle paid for found no problem, but 100% of the studies paid for by non-industry sources raised questions.

NEUROTOXINS AS A FOOD ADDITIVE

The manifestations of aspartame disease in young children are myriad. They include severe headache, convulsions, unexplained visual loss, rashes, asthma, gastrointestinal problems, obesity, marked weight loss, hypoglycemia, diabetes, addiction (probably largely due to the methyl alcohol), hyperthyroidism, and a host of neuropsychiatric features. The latter include extreme fatigue, irritability, hyperactivity, depression, antisocial behavior (including suicide), poor school performance, the deterioration of intelligence, and brain tumors.

An average aspartame-sweetened beverage would have a conservative aspartame content of about 555 mg/liter, and therefore, a methanol equivalent of 56 mg/liter (56 ppm). For example, if a 25 kg child consumed on a warm day, after exercising, two-thirds of a two-liter bottle of soft drink sweetened with aspartame, that child would be consuming over 732 mg of aspartame (29 mg/kg). This alone exceeds what the FDA considers the 99+-percentile daily consumption level of aspartame. The child would also absorb over 70mg of methanol from that soft drink. This is almost ten times the Environmental Protection Agency's recommended daily limit of consumption for methanol.

To look at the issue from another perspective, the literature reveals death from consumption of the equivalent of 6 gm of methanol. It would take 200 12 oz. cans of soda to yield the lethal equivalent of 6 gm of methanol. According to FDA regulations, compounds added to foods that are found to cause some adverse health effect at a particular usage level are actually permitted in foods only at much lower levels. The FDA has established these requirements so that an adequate margin of safety exists to protect particularly sensitive people and heavy consumers of the chemical. Section 170.22 of Title 21 of the Code of Federal Regulations mandates that this margin of safety is 100-fold below the "highest no-effect" level. If death has been caused by the methanol equivalent of 200 12 oz. cans of aspartame sweetened soda, one hundredth of that level would be two cans of soda. The relationship of the lethal dose to the "highest no effect" level has tragically not been determined for methanol but assuming very conservatively that the level is one hundredth of the lethal dose, the FDA regulations should have limited consumption to approximately 24 ounces of aspartame-sweetened soft drink per day.

The high ethanol/methanol ratio of alcoholic beverages must have a very significant protective effect given that ethanol antidotes methanol, so ignore the argument that methanol already exists in alcoholic beverages without untoward effects. This is absurd given that alcoholics have a much higher incidence of cancer and other degenerative diseases, none of which can be attributed to ethanol alone. In aspartame, the methanol is released, once in the body, unfettered by ethanol to be a pure poison.

The FDA allows a lower safety margin only when "evidence is submitted which justifies use of a different safety factor." (21.C.F.R.170.22) No such evidence has been submitted to the FDA for methanol. Thus, not only have the FDA's requirements for acute toxicity not been met, but also, no demonstration of chronic safety has been made. The fact that methyl alcohol appears in other natural food products does not exonerate its presence in aspartame, but increases greatly the danger of chronic toxicity developing by adding another unnatural source of this dangerous cumulative toxin to the food system.

Since the amino acid phenylalanine can be neurotoxic, and can affect the synthesis of inhibitory monoamine neurotransmitters, the phenylalanine in aspartame can mediate neurologic effects.

Chemicals and compounds that affect physiological systems are classified as drugs by the Food and Drug Administration (FDA), and are subject to considerably more demanding regulatory procedures than food constituents. Moreover, because food additives must be shown to be physiologically inert in order to win initial FDA approval, once they have obtained this approval they are exempt from the requirement, imposed on all drugs, that their safety be continuously monitored. Companies that manufacture and use approved food additives are not obligated to monitor adverse reactions associated with consumption of their product, nor to submit to the FDA reports of such adverse reactions; they are also not required to carry out further government-mandated research programs to affirm their product's safety.

However, the consumption of a number of food additives can cause physiological effects, which include, for some, modification of the chemical composition and functional activities of the nervous system.^{1, 2} Moreover, in the case of aspartame these neural effects were largely unexplored prior to the compound's addition to the food supply, and were not a factor in calculating the quantities that individuals can safely consume (the ADI, or acceptable daily intake, currently set for aspartame at 50 mg/kg).³ The effects of aspartame, and of certain other food additives, like caffeine, involve subtler biochemical changes, as well as functional consequences that are demonstrable only in specially treated animals⁴ (and possibly, by extrapolation, only in especially vulnerable people).

Although these physiological effects are unrelated to the reason that aspartame was placed in food, they have important health implications given the very large number of people who consume aspartame. If only 1% of the 100,000,000 Americans thought to consume aspartame ever exceed the sweetener's ADI, and if only 1% of this group happen coincidentally to have an underlying disease that makes their brains vulnerable to the effects of an aspartame-induced rise in brain phenylalanine levels, then the number of people who might manifest adverse brain reactions attributable to aspartame would

still be about 10,000, a number on the same order as the number of neurologically related consumer complaints already registered with the FDA and other federal agencies.^{5, 6}

Doses of aspartame, which are within the range actually consumed by some people can affect the chemical composition of the brain, and thereby contribute to particular CNS side effects, including headaches,⁷ inappropriate behavior responses,^{8, 9} and seizures.^{10, 11}

The major bio-chemical effect of aspartame, in humans, is to raise blood and, presumably, brain phenylalanine levels¹²; in contrast, its main effect in rodents is to raise blood (and brain) tyrosine levels,^{13, 14} and tyrosine is often the antidote to phenylalanine's effects on the brain. This species difference makes questionable the extrapolation of much of the rodent literature to humans.

The existence of this major metabolic difference between rodents and people underscores the point that only large-scale human studies could determine whether or not aspartame is risk-free. But aspartame cannot be shown to be risk-free, and its regulatory classification should be changed, for example, to that of a drug.

The Effect of Aspartame on Brain Phenylalanine Levels

The consumption of an aspartame-laden food or beverage contributes to the plasma the three natural compounds contained within the aspartame molecule: the amino acids phenylalanine and aspartic acid, and the alcohol methanol,¹⁵ as well as various peptides (like B-aspartame or the aspartyl-phenylalanine diketopiperazine that are formed from it spontaneously, on the shelf, or enzymatically, after its consumption).

Plasma phenylalanine levels are not regulated by any known homeostatic mechanism. At any particular time plasma levels simply reflect the amounts of phenylalanine being absorbed from the foods most recently eaten.^{16, 17} Consumption of the ADI aspartame dose is thus able to elevate plasma phenylalanine levels about threefold.¹⁸

Consumption of dietary phenylalanine in the usual way, as a constituent of protein, does not elevate brain phenylalanine levels.¹⁹ This is because the protein elevates plasma levels of the other large neutral amino acids (LNAA) (valine, leucine, isoleucine, tryptophan, tyrosine) more than those of phenylalanine. These other amino acids are considerably more abundant than phenylalanine in the protein, and the branched-chain amino acids, unlike phenylalanine, are largely unmetabolized when they pass through the portal circulation.²⁰

In contrast, consumption of phenylalanine in the form of aspartame, with the other LNAA, that are always present in proteins, elevates plasma phenylalanine levels without elevating those of the other LNAA, this causes marked elevations in the plasma phenylalanine ratio (the ratio of the plasma phenylalanine concentration to the summed concentrations of the other LNAA).¹³ Aspartame is the only known phenylalanine-containing food that elevates this ratio.

An elevation in the plasma phenylalanine ratio causes a parallel rise in brain phenylalanine levels, since a single transport macromolecule within the endothelial cells lining the brain's capillaries mediates the uptake of all of the LNAA; this macromolecule is unsaturated at normal plasma LNAA levels; and each of the LNAA's compete for attachment to it, their success depending on their relative affinities for it and their plasma concentration relative to those of its competitor.^{4, 21} The elevation in the plasma phenylalanine ratio also tends to reduce the corresponding ratios for the LNAA, thus decreasing their brain uptakes and tending to lower their brain levels.¹³ [Aspartame fails to lower brain tyrosine levels in the rat because the rat's liver hydroxylates dietary phenylalanine so rapidly that plasma tyrosine levels rise even more than those of plasma phenylalanine.^{13, 14} However, in humans dietary aspartame probably reduces brain tyrosine uptake.]

If an aspartame-containing beverage is consumed along with, for example, a carbohydrate-rich, protein-poor dessert food, its effect on brain phenylalanine is doubled.¹³ This is because the insulin secretion elicited by the carbohydrate selectively lowers plasma levels of the branched-chain amino acids (by facilitating their uptake into skeletal muscle), without having much of an effect on plasma phenylalanine; this increases the effect of the aspartame on the plasma phenylalanine ratio.¹⁷ A similar doubling may occur if the eater happens to be one of the perhaps 10 million Americans who are, without knowing it, heterozygous for the phenylketonuria (PKU) gene.²²

Once within brain, neurons producing certain neurotransmitters, such as dopaminergic nigrostriatal cells, the excess phenylalanine can inhibit enzymes (like tyrosine hydroxylase) needed to synthesize the neurotransmitters. Excess circulating phenylalanine can also diminish the production of brain catecholamines and serotonin by competing with their precursor amino acids for transport across

the blood-brain barrier. Hence, physiological processes that depend on the sustained release of adequate quantities of these transmitters can be affected.

One such process creates greater sensitivity to seizures.²³ In humans, aspartame, regardless of dose, causes greater increases in plasma (and brain) phenylalanine than tyrosine. (As shown below, sufficiently high aspartame doses, which transiently exceed the liver's capacity to hydroxylate phenylalanine, can also potentiate seizures in rodents, whether these seizures are generated by drugs, electroshock, or inhalation of fluorothyl.)

All of these relationships have now been demonstrated; most recently, the ability of phenylalanine to suppress dopamine release.²⁴

Aspartame and Seizure Susceptibility

To determine whether aspartame intake could modify seizure susceptibility, perhaps by increasing plasma and brain phenylalanine levels, one of our group has examined its effects on the incidence of seizures, their speed of onset, and the amount of convulsant required to produce the seizures among mice given treatments known to be epileptogenic.²⁵ In general, animals received various aspartame doses 1 hr before a CD50 dose of the seizure-inducing treatment, or a fixed aspartame dose 1 hr before various doses of the treatment. The number of animals in each treatment group exhibiting seizures in the next 60 minutes were counted (when the treatment was pentylenetetrazole), or the time passing until a given animal had a seizure (when the treatment was inhaled fluorothyl or electroshock). The aspartame doses used were those shown, in the mice, to cause blood phenylalanine levels to rise by at least as much as blood tyrosine, i.e., doses of 1000 mg/kg or greater.

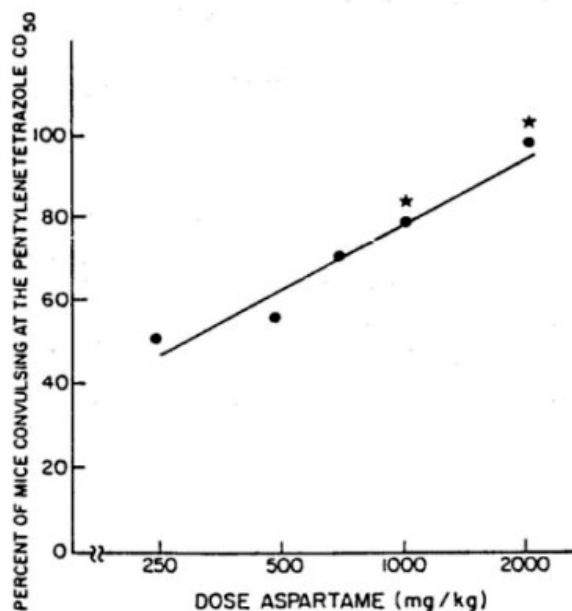


Figure 2. Effect of aspartame pretreatment on the percentage of mice convulsing following the administration of the CD50 dose of pentylenetetrazole.

Groups of male CD-1 mice (average $n = 24$) received 0-2000 mg/kg aspartame via oral intubation followed by an SC injection of pentylenetetrazole 1 hr later. The number of mice convulsing with the various aspartame doses was determined. $p < 0.05$, significantly different from 0 mg/kg as determined by the chi-square test.

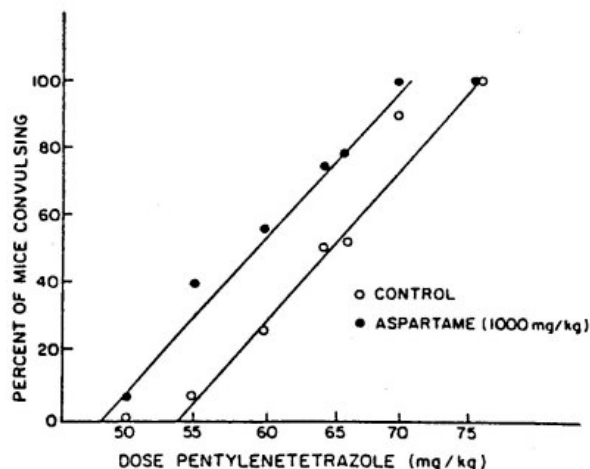


Figure 3. Effect of aspartame (1000 mg/kg) on the percentage of mice convulsing at various doses of pentylenetetrazole.

Groups (average $n = 24$) of male CD-1 mice received water or 1000 mg/kg aspartame via oral intubation followed by various doses of pentylenetetrazole, 1 hr later. The number of animals convulsing was determined. Aspartame pretreatment significantly ($p < 0.05$) shifted the dose-response curve as determined by the method of Litchfield and Wilcoxon.

Aspartame administration produced a dose-dependent increase in seizure frequency among animals subsequently receiving the CD50 dose of pentylenetetrazole (PTZ) (65 mg/kg) (Fig. 2). At the 1000 and 2000 mg/kg aspartame doses, 78 and 100% of the animals experienced seizures, compared with 50% in the water-pretreated group. Other mice pretreated with a fixed dose (1000 mg/kg) of aspartame, or with water, and given various doses (50-75 mg/kg) of PTZ an hour later exhibited a significant leftward shift of the PTZ dose response curve (Fig. 3). Enhanced susceptibility to PTZ-induced seizures was also observed among mice pretreated with phenylalanine (in doses equimolar to effective aspartame doses), but not among animals pre-treated with aspartic acid or methanol. Co-administration with aspartame of the LNAA valine, which competes with phenylalanine for passage across the blood-brain barrier,^{4, 21} protected mice from the seizure-promoting effects of the sweetener; in contrast, alanine, an amino acid which does not compete with phenylalanine for brain uptake, failed to attenuate aspartame's effect on PTZ-induced seizures.

The evidence does not indicate that aspartame itself causes seizures; but rather that it promotes seizures in animals that are already at risk (that is, animals treated with PTZ, fluoroethyl, or electroshock). In a similar manner, it is possible that doses of the sweetener that cause a sufficient increase in brain phenylalanine might increase seizure frequency among susceptible humans, or might allow seizures to occur in people who are vulnerable but without prior episodes.

It is unfortunate but perhaps not surprising that questions about aspartame's phenylalanine-mediated neurological effects arose after the sweetener was added to the food supply. New clinical data and the development of new hypotheses, based on laboratory research, can raise questions about any relatively new compound, even after that compound has passed all of the safety tests required at the time of its approval. What was and continues to be lacking is a process, free of political influence, for monitoring possible adverse reactions after food and drug additives are placed in the market.

Government-mandated safety research does not exist for politically protected chemicals and compounds, such as Thimerosal and aspartame.

CONCLUDING REMARKS

The initial approval of aspartame by the FDA in 1981 – in the face of unequivocal objections from FDA's own in-house scientists, consultants for the General Accounting Office, and even a Public Board of Inquiry – was an erroneous political and public health crime done for greed alone.

Credible scientific studies, and demographic evidence relating to the contributory role of aspartame sodas and other products in the dramatic increase of obesity, diabetes, attention deficit disorder, brain tumors and other malignancies in children.

The cover-up and obfuscation of the aspartame neurotoxin has pernicious similarities to what took place and continues to take place for Thimerosal. The introduction of aspartame into our diets took place at the exact time as the upswing in the number of vaccinations given to children, many of which contained Thimerosal. While it may always remain speculation that these two neurotoxins had a synergistic impact on the development of the current autism epidemic, it is not speculation that our Federal health agencies were co-opted to act as trade organizations for pharmaceutical corporations. This unholy alliance between state and corporation has corrupted these agencies to such an extent that they will need to be rebuilt from the ground up. In the meantime, they need to be recognized for what they are – they are not working in the public service, they have no functional oversight, they are feral, and are not looking out for the country's best interests.

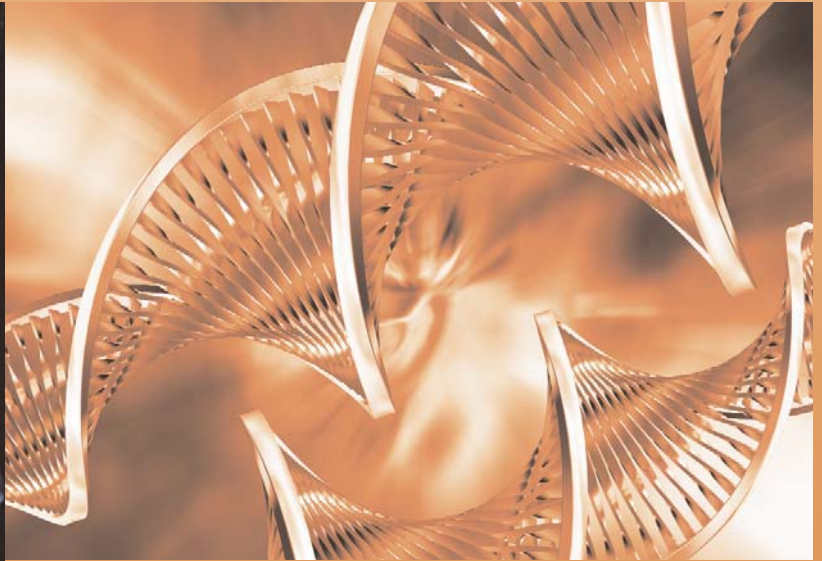
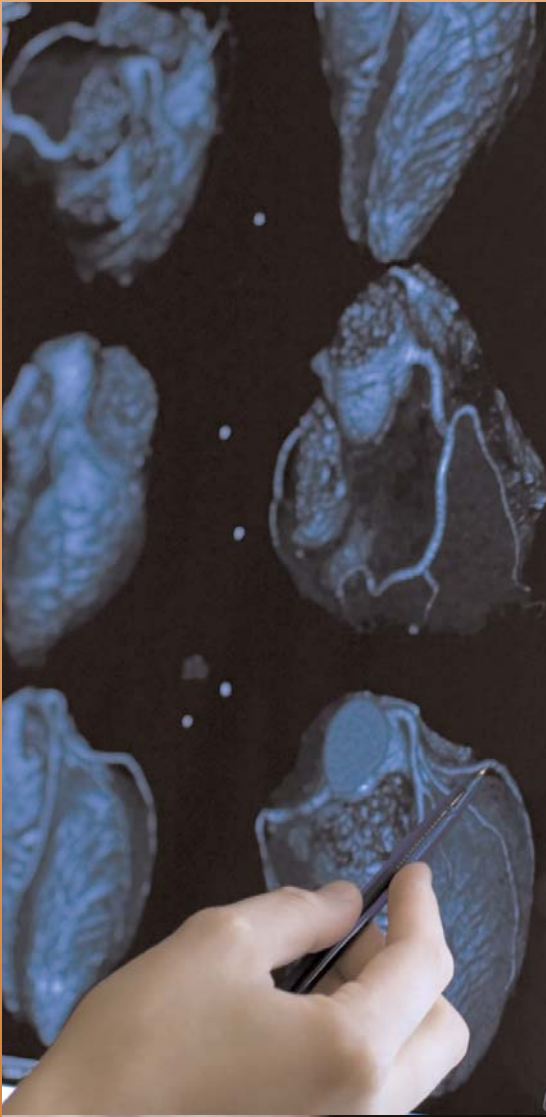
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